TRANSFER CO-EFFICIENT FOR CARBON MONOXIDE IN SPORTSMEN*

VIJAYAN, V. K., SRIKANTH, P. TRIPATHY., KUPPU RAO, K. V., AND PRABHAKAR, R

Cardio-Pulmonary Physiology Unit, Tuberculosis Research Centre, (Indian Council of Medical Research), Madras-600 031 India

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ABSTRACT

The transfer factor (TLCO) and the transfer coefficient (KCO) for carbon monoxide were measured by the single-breath method in eleven non-smoking college sportsmen. The mean transfer factor for whole lung in the college sportsmen was within normal limits when compared to predicted values of European descent; however, the transfer coefficient was high and is thought to be due to a rise in pulmonary capillary blood volume. It is postulated that continuous and prolonged training of sportsmen causes recruitment of pulmonary capillaries and this causes increased capillary blood volume even at rest.

Introduction

The most important function of the lung is to transfer gas between the air and blood and the measurement of carbon monoxide transfer (TLCO) is a direct test of its ability to do so (Davies N.J.H., 1982[1]). Bohr (1891[2]) and Haldane (Haldane and Smith, 1897[3]) believed that the lung was a secretory organ and the oxygen was actively transported across the alveolar–capillary membrane. However, it was Marie Krogh (1915[4]) who showed that oxygen was transported across the alveolar–capillary membrane by diffusion. Different methods of measurement of TLCO such as single-breath method (Ogilvie *et al.* 1957[5]), rebreathing method (Lewis *et al,* 1959[6]) steady-state method (Filley *et al.* 1954[7]) and morphometric estimates (Weibel, 1973[8]) give different results (Piiper and Scheid, 1980[9]). The transfer factor per unit alveolar volume (Transfer coeffcient or KCO) reflects the performance of the ventilated parts of the lung independent of their size (Lipscomb *et al,* 1978[10]). Because of paucity of data on single-breath diffusing capacity and transfer co-efficient in Indian literature, a study was undertaken in healthy. non-smoking college sportsmen.

Material and Methods

Eleven sportsmen of Loyola college, Madras who were active participants of various games and athletics conducted at inter-University and inter-state competitions were studied. All of them were non-smokers. A thorough clinical examination, a 12 lead electrocardiogram, oxygen consumption and simple pulmonary function tests such as Forced Vital capacity (F.V.C.), Forced Expiratory Volume in 1 second ($F.E.V_1$) and $FEV_{1\%}$ were done to exclude any respiratory or cardiovascular disease. The single breath diffusing capacity (Cotes 1975[11]) was measured on a Resparameter (P.K. Morgan Ltd. U.K.). The measurements were made with the subject sitting and a minimum of two consistent readings were obtained for each subject. The highest value obtained was used for analysis and the readings were corrected to body temperature, ambient pressure and full saturation with water vapour (BTPS). The effective alveolar volume (VA) was measured by the gas dilution method (Cotes 1975[11]) and the transfer Co-efficient (KCO) was calculated from the ratio of TLCO to VA. The measured values are considered normal, if it is within ±15% of the predicted value.

* Based on the paper presented at the Third National Congress on respiratory diseases, sponsored by Indian Chest Society, Madras-December 1983.

Results

The physical characteristics of the study subjects are given in Table-1.

TABLE–1

PHYSICAL CHARACTERISTICS OF THE SUBJECTS STUDIED

FACTOR	MEAN ± S.D
AGE (YEARS)	20.18 ± 3.14
HEIGHT (CMS)	173.27 ± 9.46
WEIGHT (KG)	58.55 ± 9.65
B.S.A (m^2)	1.70 ± 0.18

The mean age was 20.18 ± 3.14 years. the mean height 173.27 ± 9.46 cms. and the mean body surface area (B.S.A.) was 1.70 ± 0.18 m^2. Table-2 shows the basic lung function data done to exclude any respiratory impairment in these subjects.

TABLE–2
LUNG FUNCTION DATA

L.F.T.	MEAN ± S.D
V.A (L)	5.10 ± 0.99
F.V.C. (L)	4.03 ± 0.63
$F.E.V_1$ (L)	3.58 ± 0.69
$F.E.V_{1\%}$	88.80 ± 8.38

The mean effective alveolar volume (VA) by gas dilution method was 5.10±0.99 litres. The results of dynamic pulmonary function tests such as F.V.C., $F.E.V_1$ and F.E.V1 $_{1\%}$ were within normal limits, when compared to the predicted values of Indian population (Kamat *et al,* 1982[12]).

The Carbon monoxide transfer factor (TLCO) and the transfer co-efficient (KCO) for individual subjects end the mean values are given in table-3.

TABLE–3
CARBON MONOXIDE TRANSFER FACTOR (TLCO) AND TRANSFER CO-EFFICIENT (KCO)

S. No.	TLCO (m.mol/mt/ K.Pa)	TL%	KCO (m.mol/mt/ K.Pa/L)	KCO%
1.	14.3	124.2	2.77	143.5
2.	12.41	87.1	2.02	104.6
3.	16.23	149.7	2.58	135.0
4.	16.49	146.7	2.54	132.2
5.	11.96	111.2	2.81	145.5
6.	10.97	98.0	2.32	120.2
7	13.45	103.4	2.54	130.2
8.	9.68	84.1	1.97	102.0
9.	10.36	89.6	2.25	114.7
10.	9.51	86.2	2.55	132.8
11.	11.21	89.7	2.78	144.7
MEAN	12.41	107.28	2.47	127.76
± S.D.	± 2.46	± 23.48	± 0.21	± 15.36

When compared with the predicted values of males of European descent (Cotes, 1975[11]), the mean carbon monoxide transfer in those sportsmen was within normal limits. However. the mean transfer coefficient (KCO) was high.

Discussion

The elegant works of Roughton and Forster (1957[13]) had revolutionised the interpretation of the carbon monoxide transfer factor and they showed that the transfer factor was the sum of two conductances in series, that of membrane only (Dm) and that for the chemical reaction rate ($\phi_x V_c$).

$$\text{i.e. } \frac{1}{TL} = \frac{1}{DM} + \frac{1}{\phi_x V_c}$$

Where ϕ is the rate of combination of CO with haemoglobin and V_c is the pulmonary capillary blood volume that is proportional to the number of haemoglobin combining sites.

It was observed by Hamer (1963[14]), Gurtner and Fowler (1971[15]) and Lipscomb *et al* (1978[10]) that Dm changes in direct proportion to the effective alveolar volume (VA) and also that the ratio of $\frac{Dm}{VA}$ was constant at different lung volumes. Morphologically, it has been shown that there were adequate changes in alveolar surface perunit lung volume; however, there was little change in the harmonic mean barrier thickness (Gil and Weibel, 1972[16]). Weibel (1973[8]), by electron-microscopy had shown that the alveolar septum directly separating the alveolar gas from capillary blood, was the thinnest portion and a thin membrane of constant thickness is maintained between gas and blood at all lung volumes. This may be responsible for the modest changes in Dm as a function of effective alveolar volume (VA). Pulmonary capillary blood volume (V_c) remains essentially the same at all lung volumes and this is the principal cause of high TLCO/VA (KCO) at lower lung volumes.

The mean TLCO (12.41 ± 2.46 m.mol/min./kpa) in our subjects was similar to the predicted mean TLCO (11 m.mol/mt/kpa) of European descent (Cotes, 1975[11]). However, the mean KCO (2.47 ± 0.21m. mol/mt/kpa/L) was higher than the mean predicted KCO (1.9 m.mol/mt/kpa/L) (Cotes, 1975[11]) of European descent. Gupta *et al* (1984[17]) observed that the mean TLCO in Northern Indian subjects was 31.2 ± 6.4 ml/mt/mmHg (i.e. 10.45 m.mol/mt./kpa) and this value was slightly lower than the mean TLCO noticed in our subjects. Though one would expect a low TLCO compared to European descent, because of low effective alveolar volume (VA) in our subjects, the maintenance of normal TLCO in our subjects even after a proportional reduction in Dm (Lipscomb *et al.,* 1978[10]) is due to a rise in pulmonary capillary blood volume, The high capillary blood volume is indirectly reflected as high KCO in our subjects.

Cores (1975[11]) believes that there is no ethnic variation in transfer factor. He is of the opinion that the smaller lung volume is associated with a lower value for the diffusing capacity of the alveolar capillary membrane, but this is offset by the volume of blood in the alveolar capillaries being some-what larger than in Europeans. It is not clear that the observed rise in pulmonary capillary blood volume in our subjects as shown by high KCO is due to the ethnic variations or due to physical activity of the sportsmen. It may be hypothesised that the continuous and prolonged training of sportsmen result in recruitment of pulmonary capillaries, resulting in increased capillary blood volume even at rest.

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Correspondence/Request for Reprints to: Dr. V K Vijayan: ICMR Tuberculosis Research Centre, Chetput, Madras-600 031